Popular Article

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A brief overview on common salt toxicity in pig and poultry

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Although common salt is a necessary component that is included in animal feed and rations, the amount that is consumed is what renders it harmful. Toxic effects from consumingtoo much sodium chloride is known as "water deprivation syndrome." All animals, including humans and poultry, can become poisoned, although pigs and fowl are the most vulnerable.

Sources of poisoning: Feeds containing excessive amounts of common salt, accidental over-ingestion of common salt, or excessive licking of salt licks kept on the property, especially when the animals have been on restricted salt supply or after a period of salt deprivation for quite a while, can cause the animals to experience salt cravings or salt hunger. Carnivores canalso experience salt hunger by consuming excessive amounts of salty meat or brines flavoredwith meat and feeding swill to pigs. Low levels of vitamin E and amino acids including Sulphur, salt whey from cheese manufacturers or waste from salted seafood, oil fields because salt wateris an effluent from the manufacturing of oil. Salt poisoning can also occur when animals are given too much sodium sulphate or another sodium salt.

Mechanism of toxicity: The precise mechanism is unknown. But the biggest disturbances arein the sodium ions and water balance. A buildup of sodium ions in the gastrointestinal systemcan result in moderate discomfort, water secretion into the intestine's lumen, diarrhoea, and subsequent dehydration.

Clinical signs: Anorexia, extreme thirst, salivation, initially diarrhoea followed by constipation, polyuria followed by anuria, nasal discharge, and a weak pulse are all common symptoms of salt overdose. The skin and ears feel cold yet the body temperature is normal. Muscle rigidity, hyperirritability, blindness, stumbling, walking in a circle, pedaling of the limbs, recurring convulsive seizures, recumbency, coma, and death within a few hours to a fewdays are all symptoms.

In contrast to severe thirst, respiratory distress, fluid discharge from the beak, weakness,wet faeces, and limb paralysis in poultry, vomiting in dogs, profuse watery diarrhoea with colicin ruminants, diarrhoea with colic, mucus in faeces, knuckling of fetlocks, dehydration, and prostration in lactating animals are more noticeable.

Post-mortem lesions

I. Congestion and inflammation of gastrointestinal tract.
II. Faeces are fluidly and dark or dry.
III. Hydropericardium.
IV. Severe acute inflammation of gastric and intestinal lining.
V. Oedema of tissues and body cavities.
VI. Renal congestion.
VII. VII. Cerebral cortex oedema (eosinophilic meningoencephalitis in pigs; polioencephalomalacia in cattle). Pigs almost always have a considerable number of eosinophils in the dilated perivascular space and meninges, whereas chickens do not. Other abnormalities include vacuolization, disruption of the region between the cortexand white matter, and neuronal degeneration and mild general gliosis.
VIII. In mature birds, deposits of uric acid are found in the kidneys, ureters, and droppings as well as hepatic congestion in chicks and hyperaemia of the organs.
IX. Congestion of liver and kidneys and lungs are collapsed and full of blood in dogs.

Diagnosis

I. History of salt ingestion.
II. Clinical signs of poisoning including excessive thirst.
III. Post-mortem lesions.
IV. Species involved.
V. Circumstantial evidence of relatively restricted water o/salt supply.
VI. According to laboratory tests, dead animals' brain sodium levels exceeded 1800 ppm,while live animals' plasma sodium levels exceeded 150 mEq/L.

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Differential diagnosis

I. Encephalitis
II. Injury to CNS
III. Poisoning due to organophosphate compounds - but there is hypothermia.
IV. Chlorinated hydrocarbon poisoning, although there is neither thirst or excessive heat.
V. poisoning from medications or plants that stimulates the central nervous system.
VI. Poisoning due to lead or other metals where gastric symptoms are more severe.
VII. Pseudorabies

Treatment

No specific antidote is available. Do not give strong natriuretic drug.

I. Remove the toxic feed/ or water.
II. Fresh water without salt should be made accessible, although at first the animal's accessto water should be limited because a substantial intake of water will kill the animal by exacerbating cerebral oedema. As a result, provide little amounts of water more frequently, returning to the water gradually.
III. Isotonic or hypotonic salt solution intraperitoneally daily for 2-3 days.
IV. Gastrointestinal tract sedatives.
V. Sedatives to counter the CNS stimulation.

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